Some of the paper read before the Section at the present meeting will be published in our pages, and we hope next year to much more fully record for our readers the work of this Section, both as represented by its papers and their discussion.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of May 3, 1892.

The President, Dr. M. A. STARR, in the chair.

ACQUIRED MYOTONIA.

Dr. G. JACOBY presented a patient with typical phenomena of this disease. The patient, a male, with a history of chancre some few years ago, but with no further symptoms, first observed, nine weeks previous to presentation, cramps in the hand, and found that flexion of the fingers caused tonic contraction. This condition existed to a marked extent in both hands, some little time elapsing before the hands could be voluntarily opened when flexed. The shoulders were beginning to be involved in process. There were no sensory disturbances. Electrical reaction gave myotonic contraction. Mechanical reaction over the hands, forearms, and shoulders was plainly demonstrated. The patient was a cigarmaker, and the speaker put the question as to whether the disease was a professional neurosis or not? One thing certain it was not a case of Thomsen's disease, but the speaker thought that there might be a link between such cases, because he had seen a similar case of acquired myotonia develop into Thomsen's disease.

LIVE ISSUES IN NEUROLOGY.

Dr. M. A. STARR selected this title for his Inaugural Address, on taking the chair as the new President of the Society. After reviewing the work of the Society for the past few years, the speaker offered a suggestion in the mapping out of future work, it was, that there should be a more general discussion of neurological subjects. Collective investiagtion of disease was certainly of the greatest value.

For such discussion, the subject presented many divisions, especially in the cases in which the pathology was still an open question, also in the theory of disease. It should be the aim to connect symptoms with underlying lesions that the pathology would explain the disease. One of the interesting studies should be the possible relation of physiological chemical processes to the various functional nervous affections. Turning from theory to fact, would not some general discussion which would bring individual experience to a focus aid greatly in prognosis and therapeutics? It was only by mutual co-operation that the Society could be made of the greatest service, and it was with the hope that some such plan could be carried out that the speaker urged it.

The detailed histories of the three cases of angioneurotic œdema were then read.

Case I.—Female, twenty-eight years of age, with good family and personal history. She could assign no cause for the peculiar disease from which she had suffered at intervals for two years. The symptoms consisted of a stiffness, burning and swelling of the right side of the face whenever the patient exposed herself to cold. It came on sometimes spontaneously without any exciting cause. To the touch the swollen part was perceptibly harder than normal. This condition might pass off in a few hours, but had lasted three or four days. As far as discoverable the condition bore no relation with the patient's general health, indigestion or menstruation. When she first presented for examination, the right half of the face was swollen, the surface of the forehead was elevated, the tissues about the eye, and eyelids, and soft parts beneath the eye, were swollen and stiff, and so tense that the eye could not be opened as readily as the other. The cheek was perceptibly rounded and fuller than the other and the chin shared in the swelling. The neck was not involved. The swollen surface was cold and had whitish yellow tint. An application of heat for a few minutes had no perceptible effect. The face had an appearance of œdema, but did not pit on pressure. It resembled most exactly the condition seen in myxœdema. Under massage the condition subsided in a few hours. This treatment was kept up daily for two months. There had been no return of the trouble.

Case II.—Female, aged thirty-seven years, was subjected to severe emotional strain for several months before the present illness. The first symptom noticed was a swelling of the hands whenever they were placed in cold water. When winter came on this swelling was noticed in other parts of the body. Examination showed a well-nourished woman with no physical signs of disease. Her face was swollen in the lower part of both cheeks, was hard to the touch, did not pit on pressure, and waxy in appearance. The left hand was subjected to experiment. It was found that by holding the hand in cold water for one minute, swelling, distension of the veins, and increase of the temperature of the part took place, It was quite twenty-five minutes after removal before the parts returned to the previous condition. It was stated that the nails grew more rapidly now than formerly. Various kinds of treatment had been ineffectual and the patient still suffered from these symptoms in cold weather.

Case III.—Female, aged fifty-four years. The patient was a healthy woman with good history. The present condition began at Christmas, 1891, without known cause. The fingers and thumb of the left hand, the hand itself escaping, were found swollen, dark blue in color, hard to the touch, stiff in movement, and, after exposure to cold, painful. There was no anæsthesia to touch, temperature or pain, but cold was felt colder on the affected fingers. The œdema could be slightly reduced by manipulation, but not permanently. The electrical resistance was carefully measured. but was equal on both sides. There was no atheroma of the radial or other arteries. Massage was recommended.

The essential features of the disease were as follows: A swelling of the tissues affected, of an œdematous kind, with a change in their color, temperature and feel, usually without, but occasionally with, a disturbance in the sensation of the part, and an interference with the function of the parts, due to the stiffness and swelling. The swelling was usually transient, came and went rapidly after exposure to cold, but might remain permanently. It appeared to be of the nature of œdema of the corium itself rather than of the sub-dural connective tissue. The color of the part affected was usually red, but was sometimes whitish-yellow. The temperature was generally lower than on the unaffected part. The sensations felt by the patient were those of stiffness, burning, numbness, or pain, and the discomfort produced by the exposure to cold. Any part of the body might be affected by the œdema, but the hands, feet and the face were the parts most often affected. The duration of the attacks varied from a few hours to a few days. In the majority there was a complete cessation of the symptoms between the attacks. As to the nature or pathology of the disease nothing was known. Various remedies had been tried in its treatment, but none were of much avail.

OBSERVATIONS ON THE EXCRETION OF URIC ACID.

Dr. C. A. HERTER read a paper on this subject. The paper was based on an extended series of original observations upon uric acid in health and disease. The subject was treated under the following heads:

(1) The methods used in determining uric acid and urea. (2) The variations in total uric acid excretion under the influence of diet, exercise, etc. (3) The variation in total urea excretion under the influence of diet, exercise, etc. (4) The quantitative relation of uric acid and urea in health. (5) The excretion of uric acid as influenced by drugs. (6) The excretion of uric acid in disease.

It was shown (1) that the absolute quantity of uric acid excreted varied chiefly with the character of the diet, being high on a highly nitrogenous diet, and low on a diet of carbohydrates chiefly. In health the quantity of urea excreated, varied roughly with the quantity of nitrogenous food ingested. Hence, in health, both uric acid and urea might vary widely with the quantity and quality of the food. (2) That the chief clinical criterion as to whether uric acid excretion was normal, was not the absolute amount of uric acid excreted, but the ratio of the uric acid to the urea excreted. (3) That the ratio of uric acid to urea in the twenty-four hours urine of the same individual in health, was remarkably constant. (3) That this ratio in different adult individuals in health varied from $\frac{1}{45}$ to $\frac{1}{65}$.

The much-quoted views of Dr. Haig as to the relation of uric acid to disease, were criticised at some length and were shown to rest upon insufficient evidence. The relation of uric acid excretion to chorea, migraine, neurasthenia, epilepsy and paroxysmal vomiting was discussed. The general view was maintained that there was evidence to show that uric acid was not the cause of disease, but rather its effect. Excessive uric acid excretion was found in a variety of different clinical conditions, and was to be regarded as the result of different initial morbid nutritive processes.

The most important practical feature of the paper was the establishment of the fact that each individual in health, and on a mixed diet, preserved a ratio between the uric acid excreted and the urea, which showed only slight variations. In any case where this ratio was disturbed, the degree of divergence from the normal afforded a criterion of the severity of the nutritive disorder. Moreover, this knowledge gave us a reliable and scientific indication as to prognosis and the effect of treatment. A neurasthenic, for instance, who had a ratio of 1 x 30, and showed no improvement in this ratio from week to week, was not improving. But if he showed a steady change in the ratio toward the normal, we knew that he was improving, no matter what the symptoms might be at the time. We had in this ratio a far more accurate index of the patient's actual condition and progress than any that had heretofore been at our command.

Dr. L. C. GRAY said that the one fact of value elicited by Dr. Herter was the relation of uric acid to disease, but he thought that one of the drawbacks to this knowledge being of practical utility, was, that we were not by any means positive as to the normal standard of the ratio. He could not say that he had observed the same action from a nitrogenous diet as had the writer of the paper. Dr. Herter had not classified neurasthenia, but the speaker thought that the ratio in the uric acid excretion would be very different in cases of lithæmic neurasthenia.

Dr. B. SACHS thought that the paper could be used as a guide upon which to work up further facts in regard to this question of uric acid excretion in health and disease. He was satisfied that in treating a number of cases, the uric acid had been reduced by a non-nitrogenous diet and plenty of fluids.

Dr. A. D. ROCKWELL said in regard to neurasthenia, that the uric acid might or might not signify; that it was a difficult matter to say whether the disease was one of the digestive organs or a neurosis.